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Research Notes: Reaction to peanut mottle virus in plant introductions of Maturity Groups 00 through IV

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1) Reaction to peanut mottle virus in plant introductions of Maturity Groups 00 through IV.

The results of screening the soybean plant introductions in Maturity Groups II, III, and IV have been reported by Shipe et al. (1979). The results of evaluating the earlier maturity groups are reported here. Plant introductions that became available after 1976 have not been screened.

Materials and methods. The strains of peanut mottle virus (PMV) used were derived from the strain used in the previous screenings and produce similar reactions. The same inoculation methods were used.

All of the Maturity Group 00 and 0 plant introductions were evaluated in the greenhouse in 1978 and 1979, respectively. In Maturity Group I, FC 03,609 through PI 189,916 were evaluated in the greenhouse and PI 189,917 through PI 391,589A were evaluated in the field at Blacksburg. Germination was poor for some lines, so only one or two plants were available for inoculations. However, 5 to 15 plants from most lines were evaluated in the greenhouse and 15 to 30 plants in the field. Table 1 shows the plant introductions evaluated in each maturity group and those which were not evaluated because they either were missing or did not germinate.

Results. It appears that none of the plant introductions in Maturity Groups 00, 0 and I are resistant to PMV. Symptom expression varied somewhat, but none were found which were free of symptoms. A few lines, of which only one or two plants were available, did not exhibit symptoms, but it is believed they were escapes. Those lines include PI 153,237 and PI 361,121 of Group 0 and PI 86,410, PI 291,320A, PI 339,868A and PI 342,437 of Group I.

Twelve lines in Group I were observed to exhibit very mild symptoms in comparison to most other lines. Those included PI numbers 248,400, 253,658C, 291,281, 361,062A, 361,065A, 361,066A, 361,066B, 361,088A, 361,088B, 361,095, 361,098, and 361,117.

Discussion. When the results from screening the plant introductions in Maturity Groups 00 through IV are compared (Table 2), it is apparent that the frequency of resistant lines becomes successively higher in the later maturity groups. This association was noted by Shipe et al. (1979) and was assumed to

Table 1

Soybean plant introductions evaluated for reaction to PMV

Maturity Group	Highest PI no. tested	Total lines tested	PI lines not tested	
00	384,467	210	189932	
			194641	
			196502	
			196530	
0	399,074	279	FC21340	290122
			70242-4	290139
			153239	290144
			153240	297520
			153242	297522
			161989	361111
			227565	
I	391,589A	301	69507	86737
			70017	181536
			79610	196160
			79699	205085
			81033	297523
			83945-3	372404A
			86021	391583
			86133	

Table 2

Summary of frequency of plant introductions classified as resistant in Maturity Groups 00-IV

Maturity Group	Lines classed resistant	
	No.	% of total tested
00	0	0
0	0	0
I	0	0
II	7	1.3
III	16	2.5
IV	122	12.7

be either an artifact or evidence for co-evolution of the two organisms. The addition of the data for the three early maturity groups and additional verification of the data of Shipe et al. (unpublished) tend to favor the co-evolution hypothesis.

Bock and Kuhn (1975) reported the geographical distribution of PMV as southeast United States, East Africa, northeast Australia and probably Japan, West Malaysia, Venezuela and Bulgaria. These areas are largely at lower latitudes and all are within 40° of the equator. Group 00 to I soybeans are adapted outside of those limits. Assuming that the evaluated plant introductions are representative samples of the soybeans grown in their areas of adaptation, one might hypothesize some sort of association between the distribution of PMV and of lines resistant to the virus. Co-evolution is perhaps the simplest hypothesis, but more information is needed for proof. Genetic associations of PMV resistance with adaptation to specific latitudes or climates could cause similar distributions in the absence of PMV. Also, the close linkage between genes conferring resistance to PMV and soybean mosaic virus reported by Roane et al. (1980) could play a part. Unfortunately, our data don't provide much guidance as to the cause of the observed association, but the large number of lines included in the samples makes it appear to be more than a coincidence. More information on the representativeness of the germplasm collection, on the frequency of PMV resistance in later maturity groups and on the historical development of the various maturity groups might be helpful in verifying and explaining the apparent association of PMV resistance with maturity.

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